The Journal of HEREDITY

CORN SMUT SMITES VARIOUSLY
(SEE PAGE 263)

A Tetraploid Apple

Fowl Tumors

Unstable Mutant Bacilli

Antigenic Web Toes

Marie-Strumpell's Disease

The Conservation Ethic

VOLUME 37 SEPTEMBER, 1946 NUMBER 9

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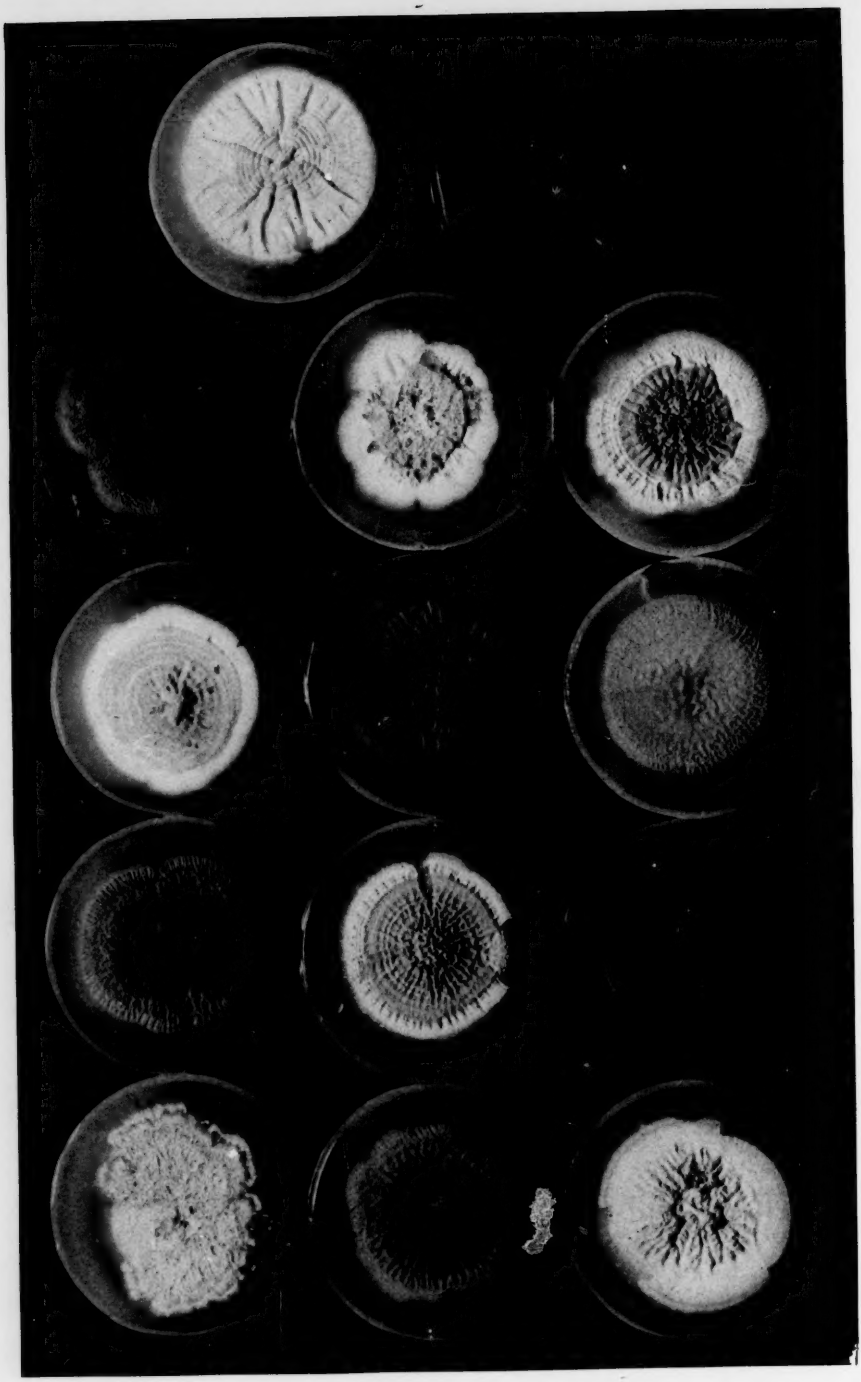


PATHOGENS HYBRIDIZE AS WELL AS MUTATE
Figure 1
(*Legend on page 259*)

PLANT PATHOLOGIST'S MERRY-GO-ROUND

A Survey of the Never-Ending Battle Against Plant Diseases

E. C. STAKMAN*

THERE are hundreds of kinds of diseases, new and old, that destroy or disfigure plants. There are rots and blights, wilts and cankers, smuts and rusts, mildews and galls, spots and scabs, each attacking particular kinds of plants. More than 3,000 different species of plant rusts are known, each with its own peculiar appearance and life habits. And there are hundreds of kinds of mildews: rose mildew, lilac mildew, oak mildew, wheat mildew, clover mildew, and so on and on. Powdery mildews alone are found on more than 1,500 kinds of plants. Every kind of plant, wild and cultivated, is subject to disease.

Plant diseases are one of the greatest hazards to successful agricultural production. The damage that they cause varies greatly with the particular kind of disease, with the variety of crop plant, and with environmental conditions. They are always a menace. And disease situations may shift and change continually.

New plant diseases may become old and old ones may become new because of changes in cropping systems, the introduction of new kinds of crop plants, and the use of new varieties. The expansion of the corn belt far northward in the Upper Mississippi Basin of the United States through the breeding of early varieties is an epic of agricultural progress. But it has increased the danger of head blight or scab on wheat and barley because the scab fungus can multiply rapidly on corn and live through the winter abundantly on cornstalks and corn stubble. Soybeans brought with them disease problems unknown in the United States before this very useful plant immigrant had become established. As new varieties of crop plants have replaced poorer ones, the relative importance of diseases often has changed also.

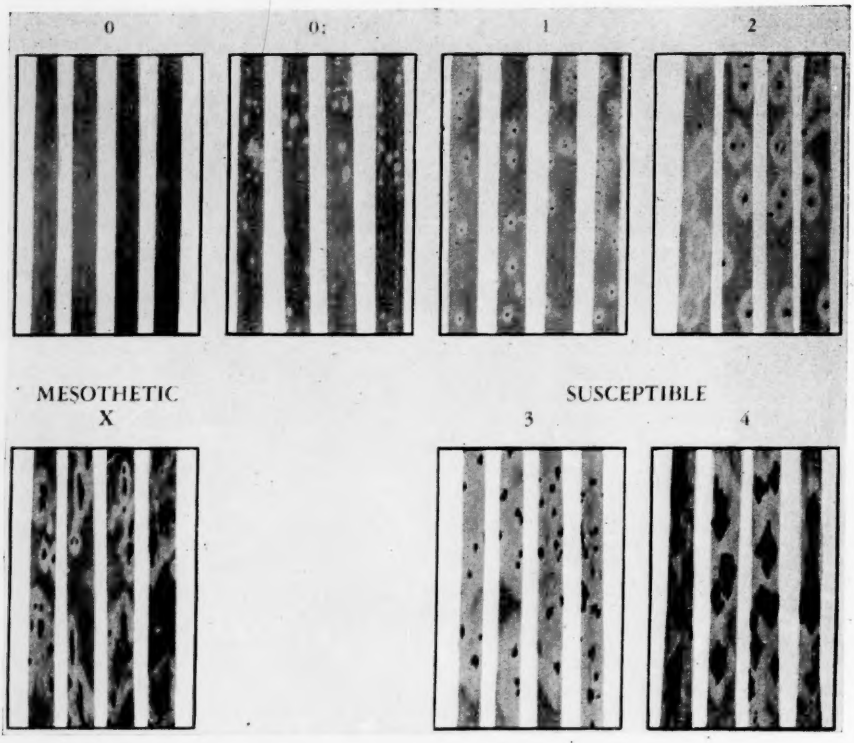
The Parade of Wheat Diseases

There have been many changes in the wheat-disease problem in the spring wheat area of the United States. Thirty-five years ago stem rust was the red terror of wheat, and stinking smut destroyed or befouled much of it unless the seed had been properly desmutted by disinfecting the seed. Head blight or scab, loose smut, and leaf rust were present but relatively unimportant.

Then came Marquis wheat to replace Preston, Haynes Bluestem, and Glyndon Fife. And farmers in those areas

*Condensation of a lecture delivered in the Spring of 1946 to many chapters of the Society of Sigma Xi.

←■ (Figure 1) This shows how new lines of corn smut can arise by hybridization. The circular discs are colonies of the corn smut fungus cultured on a suitable medium. The growth-patterns of the different lines or races of such fungi often are very characteristic. When the two haploid monosporidial lines at the top were crossed and inoculated into corn plants, millions of hybrid smut spores or chlamydospores were produced in galls formed on the plants. When these hybrid spores germinate, each normally produces a four-celled promycelium, with a sporidium or haploid segregate on each cell. Each sporidium can then be isolated with a micromanipulator and placed on nutrient agar, where it multiplies in a yeast-like or mold-like manner and produces a colony such as those in the figure. The twelve colonies shown below the two parent colonies therefore are haploid lines derived from sporidia taken from the promycelia of three of the chlamydospores resulting from the hybridization. Each of these twelve lines is different from the others in appearance, and none of them is like either parent. There are differences also in physiologic characters, sex, and virulence.



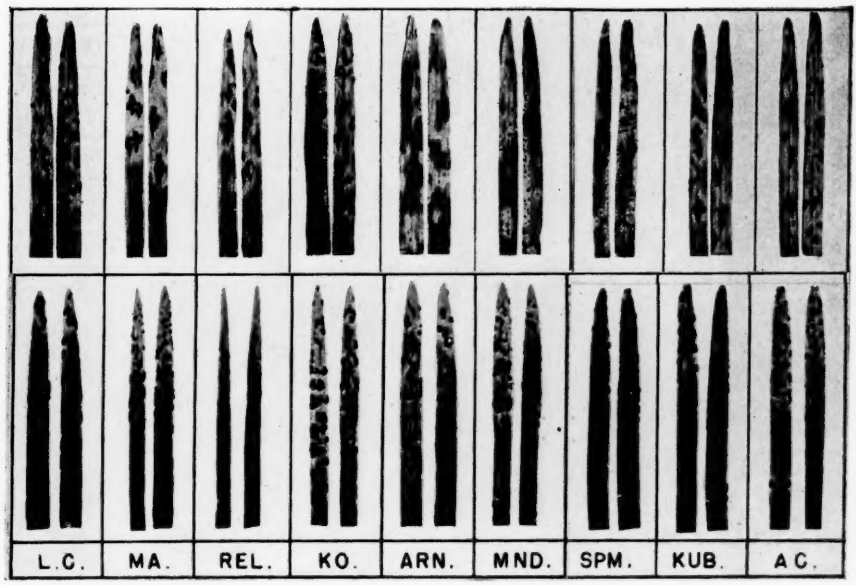
SCALE OF SUSCEPTIBILITY-RESISTANCE

Figure 2

These sections of wheat seedling leaves show the scale of resistance-susceptibility used in grading virulence of physiologic races of the wheat stem rust fungus, *Puccinia graminis tritici.* Infection types show a range from complete immunity (type zero) through high resistance (type 1) to susceptibility (types 3 and 4). The mesothetic infection type is variable, often comprising types 1 to 4 and intergrades. This is characteristic of the effect of certain rust races on certain varieties of wheat, and is not due to a mixture of races.

of the spring wheat region where wheat and corn are both grown became greatly concerned with "this new head blight that affects Marquis but not the other varieties." So uncommon had the disease been that most farmers had never noticed it. But Marquis brought it forcibly to their attention because up to 50 per cent of the heads often were partly or entirely blighted when this exceptionally susceptible variety was grown in close association with corn.

And so Marquis raised a minor disease to the rank of a major disease. At the same time it reduced a major disease, stinking smut, to the rank of a minor disease and seemed to have demoted even the dread stem rust. Marquis really was more resistant than its predecessors to stinking smut; but early ripening merely enabled it to escape stem rust damage—until the epidemic of 1916 ruined it with its companion varieties. So devastating was this epidemic that many farmers stopped growing wheat entirely and thousands of others shifted from bread wheats to durums. And so the durum era began.



DIFFERENTIAL REACTION TO TWO RACES OF STEM RUST

Figure 3

Seedling leaves of nine varieties of wheat are shown, inoculated with two races of wheat stem rust. The upper row is inoculated with race 56; the lower row with race 17. Little Club and Marquis are equally susceptible to both races. Reliance is susceptible to race 56 but immune from race 17; and Kota is susceptible to both. The three durum varieties, Arnautka, Mindum, and Spelmar, are resistant to race 56 but susceptible to race 17. Kubanka and Acme are susceptible to both. By determining the differential effects of races of stem rust on such a battery of test varieties, more than two hundred races of wheat stem rust have been identified. The sudden expansion of race 56 of the wheat stem rust caused the disastrous epidemics of 1935 and 1937 on Ceres wheat and ended the active career of this previously resistant variety in the spring-wheat area of the Upper Mississippi Valley. Fortunately, new varieties resistant to race 56 were available to replace Ceres. Such shifts in the population of races of stem rust and other pathogens mean that only by constant watchfulness to keep ahead of the trends can disastrous epidemics and crop failures be avoided.

For several years the durums seemed to have eliminated disease hazards: stem rust, stinking smut, loose smut and leaf rust, were conspicuously absent. But root rots, which often killed one third of the plants, and ergot, hitherto almost unknown in wheat, were evidence that the durums were not proof against diseases but had merely brought about an exchange of old ones for new.

The first stem-rust resistant varieties of bread wheats, such as Kota and Ceres, shuffled the ranks of diseases once more. Stinking smut returned, loose smut assumed major importance for the first time, and leaf rust became an oppressive and destructive disease. Worse still, Ceres, the best and most popular variety, was ruined by stem rust in 1935. It was therefore largely replaced by Thatcher, which was more generally resistant to stem rust, to stinking smut, and to loose smut, but probably even more susceptible than Ceres to leaf rust and scab. Now Thatcher is being replaced by still other varieties, some of which are bringing with them a new problem in the form of bacterial blight.

Even though new wheat varieties have juggled disease problems around, there

has been constant progress in reducing total losses. Better varieties and barberry eradication have brought stem rust, the greatest single menace, under at least temporary control and possibly under permanent control, unless the disease itself changes.

Changing Plant Enemies

Unfortunately, diseases themselves can change, because the fungi, bacteria and viruses that cause them can change. The fungi that cause rusts, smuts, mildews, scab of wheat and barley, flax wilt, late blight of potato, fruit rots, and many other diseases are living moldlike parasitic plants. They produce fungus seeds, or spores, by means of which they multiply and spread just as higher plants do. It is as easy to tell the difference between spores of wheat stem rust and stinking smut as between beans and peas. And spores of different kinds of rusts are as different as different kinds of beans; those of different kinds of smuts are even more different than different kinds of peas. Naturally, a microscope is needed to see the differences between spores because even the fairly large kinds are only about one one-thousandth of an inch in diameter. Except for the difference in scale, however, anyone can learn to recognize spores of stem rust as easily as kernels of wheat.

But even a good agronomist cannot always tell by looking at wheat kernels what kinds of plants they will become. Nor can a good plant pathologist tell by looking at spores of wheat stem rust what effects they will produce. It is necessary in some cases to sow the wheat seeds in the soil to see what kinds of plants they will become; and it is necessary to sow the rust spores on wheat varieties to see what effects they will produce. There are varieties and strains of wheat and there are varieties and strains of stem rust and many other fungi. The strains of fungi are usually called physiologic races or parasitic races if they look alike under the microscope but produce different physiologic and parasitic effects.

Parasitic races of stem rust of wheat were first discovered in 1916 and more than 200 are now known. To recognize them it is necessary to inoculate in the greenhouse a dozen so-called differential wheat varieties, among which are Marquis, Reliance, and Mindum. The parasitic races are designated by number rather than by Latin names.

Epidemiology of a Rust

In cooperative work with the U. S. Department of Agriculture, about 1,000 collections of rusted wheat from the States and Mexico are identified in the greenhouses at University Farm, St. Paul, Minnesota, each year. This requires planting, inoculating, and incubating about half a million plants and taking notes on them. From these physiologic race surveys, made for more than 25 years, it has been learned that certain rust races may be prevalent in one region while others are prevalent in another region in the same year and that races may have their ups and downs in different years. In 1943 race 38 made up 80 per cent of the rust in the eastern states and less than 1 per cent in the Mississippi Valley, while race 56 made up 75 per cent and race 17, 20 per cent of the rust in the Middle West and only a little of that in the East.

Race 56, found first in 1928, increased slowly in prevalence for several years, then increased suddenly and spread rapidly until it became the principal villain in the terrific rust epidemics of 1935 and 1937, when it eliminated Ceres from the ranks of resistant wheats. While race 56 was increasing, races 11, 36, and 49 decreased until now they are seldom found. Other races, for example race 38, have varied from year to year without displaying definite tendencies to increase or decrease. And race 17 was very common between 1920 and 1930, then became so rare as to be unimportant until it flared up again in 1940 and 1941.

The rust resistance of certain wheats varies with the prevalence of rust races. As an example, Marquis had less rust in the spring wheat area than the durums in 1928, because race 36 was the most

DIFFERENTIAL VIRULENCE OF CORN SMUT HYBRIDS

Figure 4

Each set of two pots of corn seedlings is inoculated with a different pair of monosporidial lines of opposite sex. The paired combination on the right is relatively weak; that at the left is somewhat more virulent; and that in the center is very virulent. These differences are only part of the complexity the plant breeder must contend with. (See Figure 3.) There are many sex groups in corn smut, and monosporidial lines, such as those shown in Figure 1, are normally unisexual. A single one alone cannot cause smut in corn; this requires combinations of lines of opposite sex.

prevalent. In 1935 and 1937, on the other hand, Marquis and Ceres were much more heavily rusted than the durums because race 56 predominated. Several of the newer wheat varieties, such as Thatcher, Newthatch, Rival, Regent, Renown, and Pilot, have so far resisted all rust races prevalent in the spring wheat region. But they are susceptible to race 15 and certain others that have been present in very small amounts. These virulent races may or may not increase to the danger point; there is no way of foretelling.

The existence of physiological races is not limited to the stem rust of wheat. There also are races of the fungi causing leaf rust of wheat, flax wilt, flax rust, pasmo disease of flax, root rots of wheat and barley, cereal smuts, potato scab, brown rot of plums, muskmelon wilt, and many others. Indeed, parasitic races have been found within species of almost all plant disease fungi that have been investigated thoroughly, and new races are continually being produced and reproduced.

Nature Breeds New Parasitic Races

New parasitic races can be produced by hybridization, for fungi also have sex. New and virulent races of stem rust are produced abundantly as a result of hybridization between existing races on barberry, the only plant on which the sexual stage of the rust develops. The eradication of rust-suscepti-

MUTANTS OF HELMINTHOSPORIUM VARY IN VIRULENCE

Figure 5

Marquis wheat inoculated with five parasitic races of *Helminthosporium sativum*. Four of these races originated as mutants of the race marked "par." Note that with respect to their effect on Marquis wheat two of the mutants (I-11 and I-19) are more virulent than the parent race, and that two of them (I-6 and I-20) are less virulent, the seedlings inoculated with race I-20 being hardly less vigorous than the noninoculated seedlings. (After J. J. Christensen)

ble barberries therefore destroys the breeding ground for rust races.

Races of smuts also hybridize commonly. More than 5,000 distinct types of the corn smut fungus have been produced in breeding experiments started with two smut races. Many new smut types have been obtained also by crossing species of oat smuts, barley smuts, sorghum smuts, and several smuts of native grasses. Smuts that are as different as tomatoes and potatoes have been crossed, and some of the hybrids have had a degree of hybrid vigor as great as that of hybrid corn. And nature breeds thousands of such hybrids; many of them are too weak to live long, but every once in a while a vicious one is born.

Even pure lines of many fungi may suddenly change and produce new types through mutation. Many mutants are weaker in parasitism than their parents, but occasional ones are more virulent. The number of new types that can be produced is astonishing even to investigators who long ago should have gotten over being astonished by the diversity and perversity of nature. Mutants may in turn cross breed, and so the process of producing new parasitic races goes on and on.

Pathogens on the March

The ways in which these parasitic races are disseminated varies with the pathogen. Man, insects, and wind are the most effective agents. Man has done himself great harm by shipping infected seed and other propagative parts of plants far and wide, often across barriers such as oceans and high mountain ranges that prevent natural spread. In such great land areas as the Mississippi Basin of the United States wind is the most effective agent of natural dissemination of many plant pathogens such as the cereal rust fungi; and it has been shown that a shower of rust spores may be deposited over an area of a quarter of a million square miles within a few days. The numerous parasitic races may therefore be disseminated widely and may become equally established in a single season if all factors operate favorably to the pathogen.

Not only are there almost countless parasitic races of hundreds of plant disease fungi, but they develop in different ways on different crop-plant varieties under different weather and soil conditions. Although important in many phases of disease control, these facts are of greatest importance in producing and

maintaining disease resistant varieties and they are used in many breeding programs. In wheat breeding programs carried on by the United States Department of Agriculture and several state agricultural experiment stations, for example, varieties and hybrids are inoculated in the field with all available parasitic races of rusts, smuts, scab, root rots and bacterial blights. And if the weather does not favor disease development, it is made to order by covering the plants with tents to increase humidity and temperature. The object is to concentrate as many local and seasonal conditions as possible into a small space and a short time—to learn not only what does happen but also what may happen to varieties in the future. It is a test of the survival of the fittest, for it is better to be disappointed with a variety in one breeding plot than on thousands of farms.

No Rest for the Breeder

Even though new varieties may have been given the severest disease tests that can be devised, there can never be a guarantee that a crop variety will retain its resistance forever, because nature may produce new enemies or new kinds of old enemies to attack it. Breeders and pathologists, however, breed more intelligently than nature and thus try to be prepared for new enemies that she may spawn. But the price of preparedness is continual research to find out what has happened, what is happening now, and what may happen in the future.

◆

A CHROMOSOMAL CHIMERA OF THE NORTHERN SPY APPLE*

JOHN EINSET, H. WESTON BLASER AND BARBARA IMHOFE

IN the course of our investigations in polyploid apples a number of "Giant sports" including large fruited forms of McIntosh, Rome, Jonathan, and Northern Spy have been examined to find if any of them had the tetraploid number of chromosomes. Determinations of chromosome number were made in somatic cells in leaf and flower buds collected from branches brought into the greenhouse in late winter and kept there several days before the fixations were made. These sports were all found to be diploid, with the exception of a large-fruited Northern Spy that had a diploid layer of cells covering the inner, tetraploid tissue.

Numerous bud variations have been described in the apple including large-fruited sports by Shamel and Pomeroy,[3] but apparently in no case has it been demonstrated that these sports were chromosomal chimeras. Although there have been many reports of periclinal chimeras in a wide variety of plants, in only a few cases have chromosomal differences in the periclines been clearly demonstrated.[1,2]

History

The original tree of this sport was noted in 1925 in a Northern Spy orchard planted by A. I. Loop of North East, Pa. The orchard had been set about 1908 with Spy trees from an unknown source. The original tree, according to Mr. H. S. Loop, has not been as vigorous as neighboring Spy trees. This may be due to the fact that it is located near a spring or wet spot in the orchard. According to Mr. Loop the tree differed from the other trees in the orchard in that the top was flatter and the angles of the branches were wider than typical Spy. The fruit, which first drew attention, was unusually large. A few apples were symmetrical, while others had large and small segments. The flesh was softer and more tender than Spy.

*Journal Paper No. 682, New York State Agricultural Experiment Station, Geneva, N. Y.

In the fall of 1930 Mr. H. S. Loop sent several fruits of this sport to the Agricultural Experiment Station at Geneva, New York, and in the spring of 1931 scion wood from the original tree was received. At that time and in the following spring a whole young apple tree was grafted over to this interesting sport. Fruit was first harvested in 1938 and the tree has had a few fruits, but never more than a light crop, in the succeeding years. In 1935 the "Giant" Spy was budded on French crab seedlings and two trees were set in the orchard in 1937. These trees have made good growth but have not reached a bearing age.

Professors Richard Wellington, G. H. Howe, and G. D. Oberle of the Department of Pomology at the Agricultural Experiment Station at Geneva all agree that the apple is definitely a Northern Spy in fruit character, especially in flavor. There is no question of its identity in their opinion. The fruit seems to average larger and may be somewhat more irregular in contour and also perhaps slightly flatter than Spy. The color of the fruit has been very similar to that of the parent with slightly finer striping. There seems little doubt that this chimera occurred as a bud mutation of the Northern Spy apple.

Cytological Investigations

Cytological examinations of a number of buds from each of the three available trees of the sport have been made and all have been found to be of the same nature. Two layers of diploid cells cover the stem tip; tetraploid cells form the central portion. The pith and most (perhaps all) of the vascular tissue of the stem is tetraploid. In young leaves the two outer layers are uniformly diploid. The extent of tetraploid tissue in the leaf is not, as yet, adequately determined. Of several thousand counts, about ten per cent are definitely tetraploid and all of them are in cells of the vascular tissues or adjacent "sheath" cells. The petiole is principally tetraploid, and the palisade and spongy mesophyll is entirely diploid. Tetraploid cells are known to occur in lateral veins, even in some of the smaller veins at some distance from the midrib. Reconstructions of leaves from sections, noting the distribution of veins and known tetraploid cells, are contemplated. These may show more clearly the pattern of tetraploid tissue in the leaf blade.

A study of the flower may prove to be even more complicated than that of the leaf. Apparently the sporogenous tissues develop from the diploid layers of cells. The pollen has been found to be monoploid and six seedlings of this apple that were examined had the diploid chromosome number.

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✦

Resistance of Wheat Varieties to Sawfly Attack

Highly significant varietal differences were established in 24 of the 28 nurseries seeded. Hollow-stemmed varieties of *T. vulgare* were generally susceptible [to sawfly attack], solid-stemmed varieties of *T. vulgare* and varieties of *T. durum* generally resistant. Varietal differences within each group were also established. The hollow-stemmed vulgare varieties suffered greatest damage under average growing conditions and least when subjected to heavy rainfall or extreme drought conditions. It is suggested that the reaction of solid-stemmed varieties is modified by rainfall, hours of sunshine, and probably other factors, the exact nature of which could not be demonstrated with the data available. Durum varieties had high resistance in wet years and variable reactions in others. The use of lattice designs increased the efficiency of the tests in a number of cases.—A. W. Platt and C. W. Farstad. *Sci. Agric.*, June 1946.

STUDIES ON THE INHERITANCE OF DOWNY MILDEW RESISTANCE

And of Other Characters in Cucumbers*

JOHN MITCHELL JENKINS, JR.†

DOWNY mildew of cucumbers is caused by the fungus *Peronoplasmopara cubensis* (B. & C. Clinton). It has long been a limiting factor in cucumber production in many parts of the United States, particularly in the humid regions along the Gulf and as far up the Atlantic Coast as the New England States. Although most commercial growers of cucumbers either dust or spray their fields with fungicides, control of the disease in this manner has not been satisfactory and losses continue to be severe.

Several varieties of cucumbers have been reported by various workers[1,3,4,5,7] as being resistant to downy mildew, but all of these varieties have had serious faults which made them unsuitable for commercial use. These faults included poor fruit color, lack of productivity, lateness, and undesirable fruit shape. Since resistant varieties were available the logical step in the search for satisfactory control of downy mildew was the use of these varieties as parents in crosses with good commercial varieties.

For the study reported herein an inbred line of Puerto Rico No. 37 was selected as the resistant parent and an inbred variety from the University of Minnesota was used as the susceptible parent. The contrasting characters of the parent varieties are listed in Table I.

In September 1940, reciprocal crosses were made between Puerto Rico No. 37 and Minnesota No. 7.36. The F_1 was grown the following spring and backcrosses were made to both parents. The F_2 and both parents were grown in the fall of 1941. The backcross plants were lost as a result of unfavorable weather conditions. The F_3 families and the parent varieties were grown in the spring of 1942 and were classified for downy mildew reaction and for other characters. By previous observation it had been found that all plants of a susceptible variety became infected with downy mildew and died. There were no "escapes." For this reason no attempt was made to inoculate the plants artificially but rows of susceptible varieties were always planted at intervals throughout the experimental plots in order to provide an abundance of spores to infect the material being studied and for use in making comparisons with other material. Plants were classified in one of ten categories representing the degree of susceptibility to downy mildew.

Seedling Resistance and Field Resistance

In 1942 an experiment was set up to determine the relation of seedling resistance to field resistance. One hundred plants each of susceptible Colorado and resistant South Carolina No. 8-63 were started in pots in the greenhouse. As soon as the plants emerged, 50 of each variety were inoculated with a spore suspension of *P. cubensis* and were kept wet for ten hours. At the end of seven days plants which showed lesions on the cotyledons were marked as susceptible and all of the plants were transplanted to the field where they were allowed to grow to maturity, or until they died as a result of downy mildew infection.

In the group of inoculated seedlings it was found that none of the plants that

*A summary of a thesis submitted to the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Doctor of Philosophy. A preliminary report of this work, which was begun in 1936, appeared in the *Journal of Heredity*.[3]

†Horticulturist in Charge, N. C. Vegetable Research Laboratory, Wilmington, N. C.

SUSCEPTIBLE SEGREGATE

Figure 6

Varieties susceptible to downy mildew show a 100 per cent mortality. These varieties have most of the characteristics desirable in garden and commercial cucumbers, most of which are lacking in resistant varieties. The problem of breeding resistant varieties thus amounts to transferring the quality of resistance to varieties excellent in all other respects.

had shown several lesions and were classed as susceptible were resistant when grown in the field, but all of those that were classed as resistant in the seedling stage were resistant when grown in the field. Seven out of eight "doubtful" plants proved to be susceptible but the other plant was resistant when tested in the field.

In the group of seedlings that was not inoculated, 88 per cent of the susceptible plants became infected as quickly as those that were inoculated, and the remainder were infected by the time the readings in the field were made. The heavy epidemic of downy mildew obtained in the seedling stage and the close association found between seedling reaction in the greenhouse and mature plant reaction in the field suggests that selection might well begin in the seedling stage in breeding for resistance to downy mildew. This procedure would save both space and labor.

The Inheritance of Resistance to Downy Mildew of Cucumbers

The reactions to downy mildew of the parents, the F_1, and the F_2 are given

TABLE I. Contrasting characters of two parent varieties of cucumbers.

Characteristics	Puerto Rico No. 37	Minnesota No. 7.36
Downy mildew reaction	Resistant	Susceptible
Spine color	White	Black
Mature fruit color	Cream	Red
Mature fruit netting	None	Heavy
Habit of growth	Indeterminate	Determinate
Rind of mature fruit	Soft	Hard

in Table II. The 52 plants of the resistant parent, when rated for resistance, fell into classes 1, 2, and 3 with a mean rating of 1.46. The 95 plants of the susceptible parent fell into classes 8, 9 and 10 with a mean rating of 9.59. The 84 F_1 plants ranged from class 4 to class 8 with a mean of 6.89. The 711 plants of the F_2 were spread over the entire range of the reaction classes with a mean rating of 5.95. Resistant and susceptible F_2 segregates are shown in Figures 6 and 7.

The F_3 lines were grown from 665 self-pollinated F_2 plants. The means of F_3 lines for downy mildew reaction are given in Table III. One F_3 line gave a mean similar to that of the resistant parent. The means of twelve F_3 lines approached closely that of the susceptible parent, and two of these twelve F_3 lines gave a range of reaction similar to that of the susceptible parent.

The results obtained suggest that it should be possible to secure downy mildew resistance with other desirable characters from a cross of this type. A logical procedure would be to inoculate the F_2 and later selfed generations in the seedling stage and thus eliminate the susceptible plants. Many F_3 lines, possibly fifty per cent of them, could be discarded in the seedling stage before transplanting to the field. The elimination of susceptible F_3 lines and susceptible plants within the lines would reduce relatively large initial numbers to the number that could be conveniently handled under field conditions. Under this procedure selection in the field might largely be based on characters other than resistance to downy mildew.

The Relation of Downy Mildew Reaction to Color of Spines, Color of Fruit, and Mature Fruit Netting

The reaction to downy mildew of 540 F_2 plants with black spines was not significantly different from that of 171 F_2 plants with white spines. When the F_2 plants were classified for downy mildew reaction according to their F_3 means the frequency distribution was substantially the same for 497 plants with black spines as for 168 plants with white spines. Chi square for independence gave a P of .70 to .80.

It was observed that all red or orange

TABLE II. Reactions to downy mildew of Puerto Rico No. 37, Minnesota No. 7.36, and the F_1 and F_2 of a cross between them.

Name	Number of plants in indicated classes of downy mildew rating										Number	Mean
	1	2	3	4	5	6	7	8	9	10		
Puerto Rico 37 (Resistant)	31	18	3								52	1.46±0.61
Minnesota 7.36 (Susceptible)								7	25	63	95	9.59±0.63
F_1 P.R. 37 × Minn. 7.36				1	11	23	18	32			85	6.89±1.01
F_2 P.R. 37 × Minn. 7.36	3	6	59	90	111	160	142	93	39	8	711	5.95±3.04

TABLE III. Means of F_3 lines for downy mildew reaction from a cross between Puerto Rico No. 37 and Minnesota No. 7.36.

Downy mildew ratings of F_2 plants	Number of F_3 means in indicated classes (Downy mildew ratings)										No. of F_3 lines	Means of F_3 means
	1	2	3	4	5	6	7	8	9	10		
1	1				1		1				3	4.33
2					2	2	2				6	6.00
3			1	16	21	19	1	1			59	5.10
4			6	30	44	6	2	2			90	4.71
5			3	5	37	32	17	6	1		101	5.76
6				7	16	44	51	20	1		139	6.46
7				7	25	44	36	15	3		130	6.28
8					17	35	28	11	2		93	6.42
9					3	10	15	5	3		36	6.86
10						1	2	3	2		8	7.75
Total	1	0	10	65	166	193	155	63	12	0	665	5.97
Puerto Rico No. 37*	34	25	5								64	1.55**
Minnesota No. 7.36*								10	15	42	67	9.48**

* Mildew ratings of individual parent plants
** Means of individual plants
$r = +.46995$ (Exceeds the 1 per cent point)

RESISTANT SEGREGATE

Figure 7

When the resistant cucumber variety Puerto Rico No. 37 is crossed with the susceptible variety Minnesota No. 7.36, the first generation hybrids are intermediate between the parents. In the second generation the plants range from highly resistant to extremely susceptible. This plant is one of the resistant F_2 segregates.

colored fruits had black spines and that all yellow or cream colored fruits had white spines. This was in agreement with results reported by Hutchins[2] and Tkachenko.[6] Examination of 700 F_2 plants showed that all of the red or orange colored fruits were netted and that none of the yellow or cream colored fruits showed any netting.

The results indicate that in this cross no significant association was obtained between reaction to downy mildew and the three closely associated characters of spine color, mature fruit color, and fruit netting.

The Relation of Downy Mildew Reaction to Spine Texture

The frequency distribution for downy mildew was not significantly different for 529 plants with few, coarse spines than for 182 F_2 plants with many, fine spines. The chi square test for independence gave probabilities of .80 to .90. When the F_2 plants were classified for downy mildew reaction according to their F_3 means the chi square test for independence of downy mildew reaction and spine texture gave probabilities of .70 to .80. The 529 coarse textured to 182 fine textured plants is a good fit to a 3 :1 ratio with P falling between .70 and .80. There appeared to be no association between texture of spines and color of spines, nor between downy mildew reaction and spine texture or colo

The Relation of Downy Mildew Reaction to Habit of Growth

The frequency distribution of 521 determinate and 190 indeterminate F_2 plants in classes for downy mildew reaction indicated that the determinate plants tended to be more susceptible to downy mildew than the indeterminate plants. The chi square test for independence gave a P value of less than .01, that the frequency distribution was the same for the two types of plants. A similar frequency distribution was obtained for the F_3 means for downy mildew reaction of determinate and indeterminate plants. The F_2 segregation of 521 determinate to 190 indeterminate plants is a good fit to a 3:1 ratio.

The Relation of Mature Fruit Color to Hardness of Rind

In 1942 four hundred plants in an F_2 population were classified for degree of hardness of rind. It was found that 217 of the red or orange fruited plants had rinds which were classified as hard and 78 had rinds which were recorded as intermediate. Eighty-three of the yellow or cream colored fruits had rinds which were classed as intermediate and 22 of them had rinds which were as soft as the rinds of the cream colored parent. The figures obtained gave a good fit to a 9:3:3:1 ratio.

Since it was hard to distinguish red colored fruits from orange these two colors were grouped together, but upon examining the data it was found that approximately one-fourth of the red or orange fruited individuals had intermediate rinds and approximately three-three-fourths of the yellow or cream colored plants had intermediate rinds. This seemed to indicate that the individuals which were classed as hard were those with true red fruits whereas those with intermediate rinds were either orange or yellow as far as fruit color was concerned. The 22 plants with soft rinds would therefore be those having the homozygous recessive cream colored fruits. Since no yellow or cream colored fruits had hard rinds and no red or orange colored fruits had soft rinds the indications are that hard rind of mature fruit is closely associated with red mature fruit color and that soft rind is associated with cream mature fruit color.

Conclusions

The nature of resistance to downy mildew in cucumbers and its mode of inheritance in the cross studied suggests the desirability of using relatively large populations in the F_2 and in later generations. The populations would be inoculated in the seedling stage and the susceptible plants eliminated before time for transplanting to the field. This would tend to reduce the field populations to those individuals having some degree of resistance. Selections in the field could then be made for desirable commercial characters and for resistance to downy mildew.

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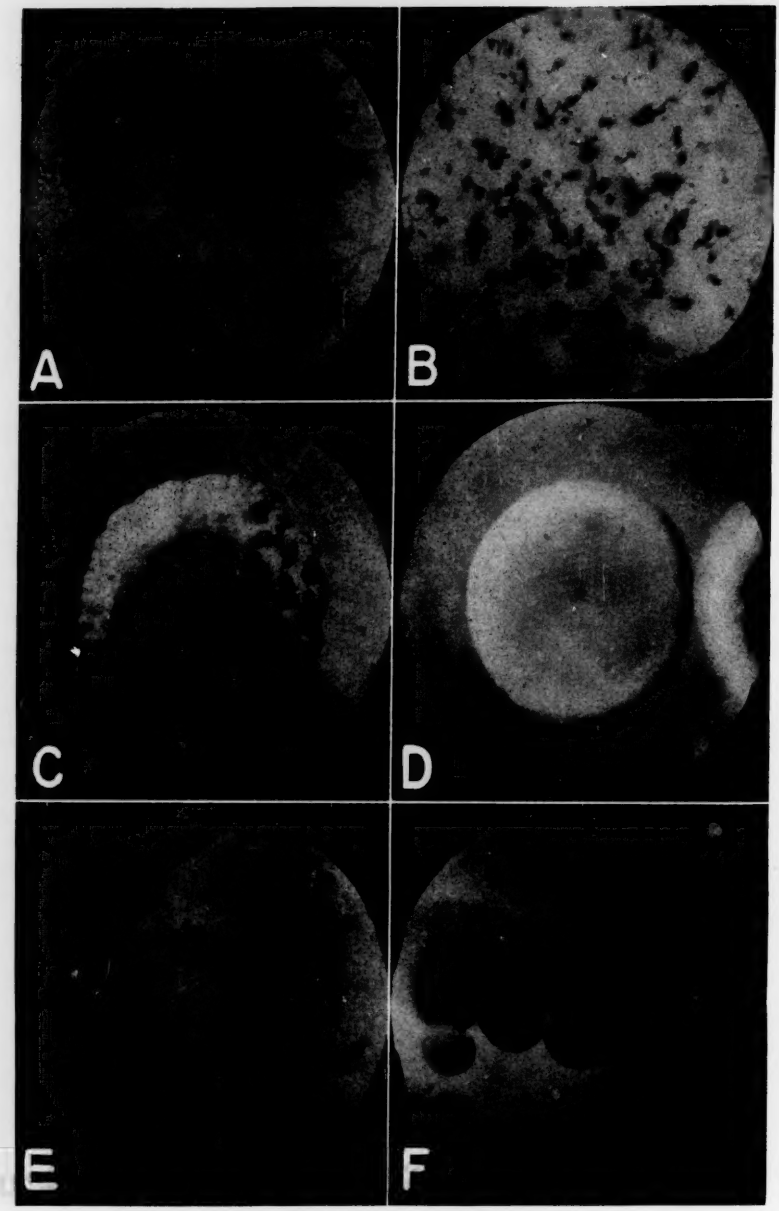

CHAINS AND RODS

Figure 8

A—"Slow fermenter" strain of *B. coli-mutabile*. Note the formation of long chains or filaments. × 400. *B*—"Prompt fermenter" strain of *B. coli-mutabile*. Note short rods typical of *Escherichia coli*. × 400. *C*—"Slow fermenter" colony on Endo agar. Note the roughness of colony × 25. *D*—"Prompt fermenter" colony on Endo agar, showing typical smooth surface of colony. × 25. *E*—"Slow fermenter" strain which has partially mutated into "prompt fermenter." × 400. Compare with *A*. Note that most of the long chains have mutated into short rods as seen in the "prompt" culture (*B*). *F*—Three days old "slow fermenter" colony on Endo agar. Note the formation of mucus or slime on the upper parts of the round colonies. × 25.

UNSTABLE STRAINS OF THE COLON BACILLUS

Two New Mutants of *B. Coli-Mutabile**

STEPHEN ZAMENHOF

FROM the PC5 strain of *B. coli-mutabile* isolated by Parr and Thomas,[4] the writer has isolated two strains which are different from the common *B. coli-mutabile* in that they do not form papillae and yet "mutate" to each other, with a very high mutability rate. In their behavior the strains resemble the "unstable variant" described by Deskowitz.[3] One of the strains is a slow lactose fermenter and forms very long chains or filaments (Figure 8*A*). These confer a characteristic rough appearance to young colonies cultured on endo (Figure 8*C*) or on eosin methylene blue agar. The other strain behaves like typical *Escherichia coli.* It is a prompt lactose fermenter. Bacteria occur as single rods (Figure 8*B*), and the colonies are smooth (Figure 8*D*).

Broth inoculated with a single "slow" chain, after 14 to 16 hours' incubation at 37°C, will show the first "prompt" single rods mutant. The proportion of these mutants increases fast: a streak culture made from broth 16 hours old shows 0.15% "prompt" cells, whereas in broth of a culture only 21 hours old "prompt" cells comprise nearly a third (30%). (Figure 8*E*.)

Observations on the mechanism of the formation of chains on a solid or semisolid agar medium reveal that the apparent slowness of multiplication of the "slow" strain has a simple physiological basis. It appears that in the chain the central cells are handicapped in reproduction, and only the end cells divide at a normal rate. On streak cultures each "slow" colony is formed by one chain whereas each "prompt" colony arises from a single cell; as a consequence of the handicapping of most of the cells in a chain, on incubation and periodical streaking "slow" colonies will increase in number much more slowly than "prompt" colonies.

Mutation Rates

Taking the above into consideration and observing the number of "slow" chains present at the moment of appearance of the first "fast" cell, the mutation rate has been estimated at 1 per 1,000 to 1 per 5,000 individual cell divisions.[5]

The above phenomena occur also when pure "slow" chains are streaked on agar. Here, too, one single "slow" chain gives rise to a colony, which, when 24 hours old, emulsified and restreaked, will show a proportion of 5 to 15 "prompt" colonies per 100 "slow" ones. In a "slow" colony the prompt cells do not form papillae but are mixed uniformly with "slow" chains; in an old "slow" colony the "prompt mutants" and their descendants confer so much of the "prompt" colony characteristics that only the *young* "slow" colonies can be discerned from pure "prompt" ones. This change in appearance refers to endo or eosin methylene blue agar only, because on plain nutrient agar the "slow" and the "prompt" colonies always look alike, although, of course, the above described "mutational processes" take place here, too. These constantly recurring mutations make it impossible to keep pure stock colonies of "slow" on agar or broth; the only way the "slow" can be kept pure is in very high dilution, in *pure* water, in the refrigerator. Any traces of food which start cell divisions will inevitably release these mutational processes and contaminate the "slow" culture with "prompt" cells.

*Paper presented at the Thirty-First Meeting of the New York City Branch of the Society of American Bacteriologists on December 28, 1944.

The "prompt" strain also "mutates" back to "slow," at a rate probably not greatly different from that of "slow" to "prompt." These mutations are, however, much more difficult to detect due to the above described apparent fast multiplication rate of the "prompt." For example, one week old "prompt" broth on streaking will reveal one "slow" colony per 300 to 5,000 "prompt" ones.

Alkalinization

Upon incubation at room temperature prolonged by one or two days, on endo plates, both "slow" and "prompt" strains undergo an interesting variation: the colonies in one spot become exceedingly mucoid and slimy (Figure 8*F*). At the same time these spots become strongly alkaline; the alkali diffuses throughout the plate whereby the entire plate becomes mucoid and decolorized. In this stage the "slow" chains break into individual *encapsulated* rods and become similar to "prompt." This variation is not a "mutation" because it is not hereditary. "Prompt" slime when emulsified in water and restreaked on fresh endo plates will always give rise to the original non-mucoid "prompt" colonies. "Slow" slime, although seemingly identical with "prompt," will give rise to original non-mucoid "slow" colonies.

Each of the "fast" and "slow" strains can be further subdivided into two strains with respect to formation of these mucoid spots. The cells of one strain will start to produce alkali and mucus after an incubation period. The cells of the other strains will never start to produce alkali even after prolonged incubation. These strains may be induced to become mucoid and to produce alkali by diffusing into the colonies alkali produced by the first strain, or by any other alkali-producing cells. This reaction can even be induced by diffusion of chemically pure sodium hydroxide into non-mucoid colonies. Furthermore, several other strains of *B. coli-mutabile* were found to show the peculiarity of producing alkali when this process is induced by exposure to a diffusing alkaline medium. Only the two strains described above show the peculiar behavior of becoming mucoid at the same time.

The production of alkali by cells exposed to alkali has been found previously by Chiari and Loeffler.[1] These authors believed that the diffusing alkali contained a peculiar enzyme or a phage responsible for the reaction. The above described experiments of the writer indicate that even as simple an alkali as sodium hydroxide has the same effect. This would appear to preclude an introduced enzyme as causing the reaction. However, a latent enzyme might be activated by the alkaline medium.

In those cases where "prompt" colonies do not become mucoid on prolonged incubation, the periphery of the colonies becomes colorless and contains many chains. These changes, however, are also not hereditary and upon restreaking on fresh endo plates the variants give rise to the original "prompt" colonies.

The above study is an example of an interwoven pattern of non-hereditary variations and of hereditary changes ("mutations"). The hereditary changes seem to reveal an instability of the "genic material" which is assumed to control the development of bacteria as of higher organisms. This has led Deskowitz[3] and other writers to stress an analogy between "mutations" in bacteria and the behavior of unstable genes in higher organisms.[2,6] The author would hesitate to call this analogy justified, as long as we do not know definitely what the bacterial "mutations" are.

Summary

The writer has isolated two unstable strains of *Bacterium coli-mutabile* which "mutate" to each other with a high frequency but without forming papillae. One of the strains is a slow lactose fermenter and forms long chains. The other behaves like typical *Escherichia coli.* Taking into consideration the effect of chain formation, the "mutation rate" has been estimated at 1 per 1,000 to 5,000 individual cell devisions.

Further study reveals also non-hereditary variations, such as mucus, capsule,

and alkali formation, all induced by alkali engendered by other cells. These variations are interwoven in a rather complicated pattern with the above-mentioned hereditary changes ("mutations").

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✦

RACIAL WISDOM AND CONSERVATION

AS I see it, the person who has developed some control of his greed, his vanity, and his fears; who has developed to the limit of his brain the accumulated understanding of man and the universe and who thinks in terms of his fellowman —that is, the human race—not for the day, for tomorrow, or even the next hundred years, but for the future at least as long as our human past; and who at the same time uses all of his influence, without violence or coercion, to prevail on his fellowman to follow his example, that individual is entitled to the connotation 'wise.'— A. J. CARLSON.

We people of the United States, on any particular day, are a segment of this species of ours in process and in progress from yesterday to tomorrow. Only if we can become wise in the sense of Dr. Carlson's definition can there be any general constructive interest in eugenics and the conservation of our race.

The development of such a broadly conceived, multi-dimensional outlook must precede any awakening of a popular interest in eugenics. Galton conceived that the motivation of eugenics must at least in part be an attitude akin to the religious. One major obstacle to the promulgation of such a new faith has perhaps been the lack of a scheme of concepts and relationships which could form its background. The basis for such a new ethic has never been very clearly outlined. For this ethic must base on the acceptance of the facts of man's ecology—his relation to his follows and to his total environment, in nature and in the social matrix of his unique species.

A novel approach to this aspect of the problem is to be found in a discussion of "The Conservation Ethic," by Professor Aldo Leopold.* The "Conservation Ethic" is envisioned as an emerging stage in the evolution of ethical concepts. With increasing socialization and interdependence, there are progressive shifts in areas that have applied to them the concept of "property." At one time in this evolution, slaves are property to be exploited at the will of the owner. Later, human beings have been removed from this category. This stage we have now reached, at least in theory. A next step could be to stop the exploitation of the still unborn members of our race. It is a popular wisecrack to ask "what has posterity ever done for us?" If we turn this around and ask what are we doing to posterity, and if we face that question squarely, it it not very funny. Asking posterity to pay for our wars is only a small part of a heedless campaign of exploitation that makes the sorry story of our treatment of our lands and forests seem positively benign. The Alhambra, says Professor Leopold, is being remodelled with a steam shovel. And the temple of life is to be remodeled with festooned

*First published in the *Journal of Forestry* (Vol. XXXI, No. 6, Oct. 1933), Dr. Leopold's remarks have recently been reprinted in a news-letter of the Pan American Section of the International Committee for Bird Preservation, 1000 Fifth Avenue, New York City. Except that Dr. Leopold does not mention The Bomb, most of his remarks might have been written yesterday.

chain reactions. This horrid prospect is so frightening that even the threat may produce a stampede as destructive as the dissolving atomic nuclei. And a stampede, says Professor Leopold, is the action of organisms which have centered their attention too closely on limited features of the environment. But let him tell his story in his own words:

When god-like Odysseus returned from the wars in Troy, he hanged all on one rope some dozen slave-girls of his household whom he suspected of misbehavior during his absence.

This hanging involved no question of propriety, much less of justice. The girls were property. The disposal of property was then, as now, a matter of expediency, not of right and wrong.

Criteria of right and wrong were not lacking from Odysseus' Greece: witness the fidelity of his wife through the long years before at last his black-prowed galleys clove the wine-dark seas for home. The ethical structure of that day covered wives, but had not yet been extended to human chattels. During the three thousand years which have since elapsed, ethical criteria have been extended to many fields of conduct, with corresponding shrinkages in those judged by expediency only.

This extension of ethics, so far studied only by philosophers, is actually a process in ecological evolution. Its sequences may be described in biological as well as philosophical terms. An ethic, biologically, is a limitation on freedom of action in the struggle for existence. An ethic, philosophically, is a differentiation of social from anti-social conduct. These are two definitions of one thing. The thing has its origin in the tendency of interdependent individuals or societies to evolve modes of cooperation. The biologist calls these symbioses. Man elaborated certain symbioses called politics and economics. Like their simpler biological antecedents, they enable individuals or groups to exploit each other in an orderly way. Their first yardstick was expediency.

* * * *

At a certain stage of complexity, the human community found expediency-yardsticks no longer sufficient. One by one it has evolved and superimposed upon them a set of ethical yardsticks. The first ethics dealt with the relationship between individuals. The Mosaic Decalogue is an example. Later accretions dealt with the relationship between the individual and society. Christianity tries to integrate social organization to the individual.

There is as yet no ethic dealing with man's relationship to land and to the non-human animals and plants which grow upon it. Land, like Odysseus' slave-girls, is still property. The land-relation is still strictly economic, entailing privileges but not obligations.

The extension of ethics to this third element in human environment is, if we read evolution correctly, an ecological possibility. It is the third step in a sequence. The first two have already been taken. Civilized man exhibits in his own mind evidence that the third is needed. For example, his sense of right and wrong may be aroused quite as strongly by the desecration of a nearby woodlot as by a famine in China, a near-pogrom in Germany, or the murder of the slave-girls in ancient Greece. Individual thinkers since the days of Ezekial and Isaiah have asserted that the despoliation of land is not only inexpedient but wrong. Society, however, has not yet affirmed their belief. I regard the present conservation movement as the embryo of such an affirmation. I here discuss why this is, or should be, so.

Some scientists will dismiss this matter forthwith, on the ground that ecology has no relation to right and wrong. To such I reply that science, if not philosophy, should by now have made us cautious about dismissals. An ethic may be regarded as a mode of guidance for meeting ecological situations so new or intricate, or involving such deferred reactions, that the path of social expediency is not discernible to the average individual. Animal instincts are just this. Ethics are possibly a kind of advanced social instinct in-the-making.

Whatever the merits of this analogy, no ecologist can deny that our land-relation involves penalties and rewards which the individual does not see, and needs modes of guidance which do not yet exist. Call these what you will, science cannot escape its part in forming them.

Ecology—Its Role in History

A harmonious relation to land is more in-

tricate, and of more consequence to civilization, than the historians of its progress seem to realize. Civilization is not, as they often assume, the enslavement of a stable and constant earth. It is a state of *mutual and interdependent cooperation* between human animals, other animals, plants, and soils, which may be disrupted at any moment by the failure of any of them. Land-despoliation has evicted nations, and can on occasion do it again. As long as six virgin continents awaited the plow, this was perhaps no tragic matter,—eviction from one piece of soil could be recouped by despoiling another. But there are now wars and rumors of wars which forestall the impending saturation of the earth's best soils and climates. It thus becomes a matter of some importance, at least to ourselves, that our dominion, once gained, be self-perpetuating rather than self-destructive.

This instability of our land-relation calls for example. I will sketch a single aspect of it: the plant succession as a factor in history.

In the years following the Revolution, three groups were contending for control of the Mississippi valley: the native Indians, the French and English traders, and American settlers. Historians wonder what would have happened if the English at Detroit had thrown a little more weight into the Indian side of those tipsy scales which decided the outcome of the Colonial migration into the cane-lands of Kentucky. Yet who ever wondered why the cane-lands, when subjected to the particular mixture of forces represented by the cow, plow, fire, and axe of the pioneer, became bluegrass? What if the plant succession inherent in this "dark and bloody ground" had, under the impact of these forces, given us some worthless sedge, shrub, or weed? Would Boone and Kenton have held out? Would there have been any overflow into Ohio? Any Louisiana Purchase? Any transcontinental union of new states? Any Civil War? Any machine age? Any depression? The subsequent drama of American history, here and elsewhere, hung in large degree on the reaction of particular soils to the impact of particular forces exerted by a particular kind and degree of human occupation. No statesman-biologist selected those forces, nor foresaw their effects. That chain of events which on the Fourth of July we call our National Destiny hung on a "fortuitous concourse of elements," the interplay of which we now dimly decipher *by hindsight only*.

Contrast Kentucky with what hindsight tells us about the Southwest. The impact of occupancy here brought no bluegrass, nor other plant fitted to withstand the bumps and buffetings of misuse. Most of these soils, when grazed, reverted through a successive series of more and more worthless grasses, shrubs, and weeds to a condition of unstable equilibrium. Each recession of plant types bred erosion; each increment to erosion bred a further recession of plants. The result today is a progressive and mutual deterioration, not only of plants and soils, but of the animal community subsisting thereon. The early settlers did not expect this, on the cienegas of central New Mexico some even cut artificial gullies to hasten it. So subtle has been its progress that few people know anything about it. It is not discussed at polite teatables or go-getting luncheon clubs, but only in the arid halls of science.

All civilization seems to have been conditioned upon whether the plant succession, under the impact of occupancy, gave a stable and habitable assortment of vegetative types, or an unstable and uninhabitable assortment. The swampy forests of Caesar's Gaul were utterly changed by human use—for the better. Moses' land of milk and honey was utterly changed—for the worse. Both changes are the unpremeditated resultant of the impact between ecological and economic forces. We now decipher these reactions retrospectively. What could possibly be more important than to foresee and control them?

We of the machine age admire ourselves for our mechanical ingenuity; we harness cars to the solar energy impounded in carboniferous forests; we fly in mechanical birds; we make the ether carry our words or even our pictures. But are these not in one sense mere parlor tricks compared with our utter ineptitude in keeping land fit to live upon? Our engineering has attained the pearly gates of a near-millennium, but our applied biology still lives in nomad's tents of the stone age. If our system of land-use happens to be self-perpetuating, we stay. If it happens to be self-destructive we move, like Abraham, to pastures new.

Do I overdraw this paradox? I think not.

Consider the transcontinental airmail which plies the skyways of the Southwest—a symbol of its final conquest. What does it see? A score of mountain valleys which were green gems of fertility when first described by Coronado, Espejo, Pattie, Abert, Sitgreaves, and Couzens. What are they now? Sandbars, wastes of cobbles and burroweed, a path for torrents. Rivers which Pattie says were clear, now muddy sewers for the wasting fertility of an empire. A "Public Domain," once a velvet carpet of rich buffalo-grass and grama, now an illimitable waste of rattlesnake-bush and tumbleweed, too impoverished to be accepted as a gift by the states within which it lies. Why? Because the ecology of this Southwest happened to be set on a hair-trigger. Because cows eat brush when the grass is gone, and thus postpone the penalties of over-utilization. Because certain grasses, when grazed too closely to bear seed-stalks, are weakened and give way to inferior grasses, and these to inferior shrubs, and these to weeds, and these to naked earth. Because rain which spatters upon vegetated soil stays clear and sinks, while rain which spatters upon devegetated soil seals its interstices with colloidal mud and hence must run away as floods, cutting the heart out of country as it goes. Are these phenomena any more difficult to foresee than the paths of stars which science deciphers without the error of a single second? Which is the more important to the permanence and welfare of civilization?

* * * *

Unforeseen ecological reactions not only make or break history in a few exceptional enterprises—they condition, circumscribe, delimit, and warp all enterprises, both economic and cultural, that pertain to land. In the cornbelt, after grazing and plowing out all the cover in the interests of "clean farming," we grew tearful about wild-life, and spent several decades passing laws for its restoration. We were like Canute commanding the tide. Only recently has research made it clear that the implements for restoration lie not in the legislature, but in the farmer's toolshed. Barbed wire and brains are doing what laws alone failed to do.

In other instances we take credit for shaking down apples which were, in all probability, ecological windfalls. In the Lake States and the Northeast lumbering, pulping, and fire accidentally created some scores of millions of acres of new second-growth. At the proper stage we find these thickets full of deer. For this we naively thank the wisdom of our game laws.

In short, the reaction of land to occupancy determines the nature and duration of civilization. In arid climates the land may be destroyed. In all climates the plant succession determines what economic activities can be supported. Their nature and intensity in turn determine not only the domestic but also the wild plant and animal life, the scenery, and the whole face of nature. We inherit the earth, but within the limits of the soil and the plant succession we also *rebuild* the earth,—without plan, without knowledge of its properties, and without understanding of the increasingly coarse and powerful tools which science has placed at our disposal. We are remodelling the Alhambra with a steam-shovel.

* * * *

I will not belabor the pipe-dream. It is no prediction, but merely an assertion that the idea of controlled environment contains colors and brushes wherewith society may some day paint a new and possibly a better picture of itself. Granted a community in which the combined beauty and utility of land determines the social status of its owner, and we will see a speedy dissolution of the economic obstacles which now beset conservation. Economic laws may be permanent, but their impact reflects what people want, which in turn reflects what they know and what they are. The economic set-up at any one moment is in some measure the result, as well as the cause, of the then prevailing standard of living. Such standards change. For example: some people discriminate against manufactured goods produced by child-labor or other anti-social processes. They have learned some of the abuses of machinery, and are willing to use their custom as a leverage for betterment. Social pressures have also been exerted to modify ecological processes which happened to be simple enough for people to understand;—witness the very effecitve boycott of bird skins for millinery ornament. We need postulate only a little further advance in ecological education to visualize the application of like pressures to other conservation problems.

For example: the lumberman who is now unable to practice forestry because the public is turning to synthetic boards may then be able to sell man-grown lumber "to keep the mountains green." Again: certain wools are produced by gutting the public domain; couldn't their competitors, who lead their sheep in greener pastures, so label their products? Must we view forever the irony of educating our sons with paper, the offal of which pollutes the rivers which they need quite as badly as books? Would not many people pay an extra penny for a "clean" newspaper? Government may some day busy itself with the legitimacy of labels used by land-industries to distinguish conservation products, rather than with the attempt to operate their lands for them.

I neither predict nor advocate these particular pressures—their wisdom or unwisdom is beyond my knowledge. I do assert that these abuses are just as real, and their correction every whit as urgent, as was the killof egrets for hats. *They differ only in the number of links composing the ecological chain of cause and effect.* In egrets there were one or two links, which the mass-mind saw, believed, and acted upon. In these others there are many links; people do not see them, nor believe us who do. The ultimate issue, in conservation as in other social problems, is whether the mass-mind *wants to* extend its powers of comprehending the world in which it lives, or, granted the desire, *has the capacity to do so.* Ortega, in his "Revolt of the Masses," has pointed the first question with devastating lucidity. The geneticists are gradually, with trepidations, coming to grips with the second. I do not know the answer to either. I simply affirm that a sufficiently enlightened society, by changing its wants and tolerances, can change the economic factors bearing on land. It can be said of nations, as of individuals: "as a man thinketh, so is he."

It may seem idle to project such imaginary elaborations of culture at a time when millions lack even the means of physical existence. Some may feel for it the same honest horror as the Senator from Michigan who lately arraigned Congress for protecting migratory birds at a time when fellow-humans lacked bread. The trouble with such deadly parallels is we can never be sure which is cause and which is effect. It is not inconceivable that the wave phenomena which have lately upset everything from banks to crimerates might be less troublesome if the human medium in which they run *readjusted its tensions.* The stampede is an attribute of animals interested solely in grass.

SOVIET GENETICS

THE authors* have presented the history and psychology which has led to the new school of genetics in the Soviet Union. They then survey the alleged scientific contributions and attempt to evaluate the claims of the non-Mendelian geneticists.

The school of genetics founded in 1935 by Lysenko and Prezent became dominant in 1940. Its basic tenets are derived from the works of Darwin, Timirjazev, Micurin and Burbank as interpreted in the light of the philosophy of dialectical materialism. "In contrast to western procedure, much of the scientific discourse of Lysenko's school is alogical, i.e., derives its conclusions not by logical argument from the facts, but by appeal to chosen authorities, by condemning views in opposition to these authorities, by analyzing the presumed states of mind of its opponents, and by estimating the value of theories by their agronomic usefulness."

Lysenko's rise to power seems to have been based upon Stalin's demand for practical results and upon political philosophy. The philosophy of dialectical materialism as developed by Marx, Engels and Lenin was made official after the October Revolution. At the All-Union Conference on Planning of Gene-

*P. S. Hudson and R. H. Richens. *The New Genetics in the Soviet Union.* Imperial Bureau of Plant Breeding and Genetics. May 1946. School of Agriculture, Cambridge, England. Price 6 shillings.

tics and Selection in 1935, a resolution was passed that genetics and plant breeding were to conform with dialectical materialism. Lysenko maintained that Mendelism and modern genetics are inconsistent with this official Marxian philosophy. Official support and liberal use of the popular press enabled him to dominate the field of genetics and plant breeding by 1940. He succeeded Vavilov as Director of the Genetics Institute of the Academy of Sciences and has been awarded many government honors.

Orthodox genetics has not been completely suppressed although it is taught at only one university. Vavilov and Karpecenko have died, but Serebrovski, Dubinin, Navasin and Zebrak are still working along Mendelian lines. However "some sort of recognition of the value of dialectical materialism in scientific work and a tendency to exalt the work of Darwin, Micurin and Timirjazev appears to characterize almost all Russian genetical publications, irrespective of the attitude taken to Lysenko's theories. . . ."

Views inconsistent with approved authority are considered "heretical." Of the various heresies, fideism, or the belief in theism, is considered particularly pernicious, and the fact that Mendel was a priest has been used to discredit his ideas. Mendelism also has been identified with the bourgeoisie and Lysenko's system with the communist society.

The new Russian "genetics" is, however, not confined to philosophical controversy. Lysenko and his followers claim to be able to induce genetic alterations by altering the environment or by graft hybridization. It is also claimed that an F_1 hybrid is always as early in maturity as the earlier parent, that biologically advantageous characters are dominant, that pure lines necessarily degenerate on selfing, and that F_1 progeny from homozygous parents may be heterozygous. Rejuvenation by crossing within pure lines is said to result in increased yields. Dolgusin goes even further and maintains that the two halves of a single plant, grown under different conditions, when crossed produce progeny with hybrid vigor. Gametic selection, or "marriage for love" also is an important aspect of Lysenko's theories. He maintains that hereditary characters are determined by the "nutritive sap" and that chromosomes have nothing to do with heredity.

The authors point out the discrepancies and fallacies in most of these claims. Statistical analysis is a "heresy" and is never used by the Lysenko school. Control experiments also are taboo. The authors criticize the non-Mendelian geneticists for their evident ignorance of elementary genetics and cytology, but suggest that some of their ideas and claims merit consideration by the western geneticists.

The analysis of the new Soviet genetics is based upon the original literature and includes the historical and psychological background as well as the biological contributions. It is evident that political philosophy has played an important part in the development of the new school of genetics in the Soviet Union. The authors suggest that Lysenko's ideas are not accepted by many of the professional workers and that the new school may not survive.

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THE OCCURRENCE OF LYMPHOID TUMORS

In Resistant and Susceptible Chickens

NELSON F. WATERS*

THE Regional Poultry Research Laboratory at East Lansing, Michigan, was established in 1938, by authority of Congress, to study the problem of viability in poultry. Since lymphomatosis was, and still is, a disease prevalent among poultry throughout the Nation, all of the facilities of this laboratory have been directed, from the beginning of work in 1939, towards its control. The opinion is justified that few flocks of chickens in the United States have escaped the ravages of this disease.

Lymphomatosis is apparently a naturally occurring malignant disease of chickens characterized by the formation of lymphoid tumors which may be found in nearly all tissues of the bird's body. Research makes it increasingly evident that this disease is communicable and may be transmitted under natural conditions, both through the egg[10] and by bird-to-bird contact.[8] It is equally evident that modifications of the environment[2,8] and genetic resistance[2,7,9] determine to a large extent the incidence of lymphomatosis in experimental groups of non-inoculated chickens when they are exposed to the naturally occurring disease.

Numerous investigators have stressed the importance of genetic selection for resistance to disease in animals. MacDowell[5] discusses the role of genetics in mouse leukemia, and Little,[4] reviewing the genetics of spontaneous tumor formation states: "there is compelling evidence that the genetic constitution of an organism plays a part in determining whether or not it will develop a tumor or tumors." In the fowl the exhaustive studies of Roberts and Card[6] on the genetics of resistance and susceptibility to pullorum disease and those of Lambert and Knox[3] for fowl typhoid demonstrate the role of inheritance in disease control in chickens.

The object of the present report is to show that genetic selection with inbreeding has decreased the incidence of naturally occurring lymphomatosis, with relation to age, in certain families within one line, while in families within another line there has been an increase.

Materials and Methods

The foundation chickens used in this study were selected from a sample obtained as eggs from nine different flocks of White Leghorns in 1939. From the original chickens a total of fourteen different inbred lines are in the formative stage of development. Representative families from two of these inbred lines, together with the tumor incidence in each generation, are shown in the pedigree charts (Figure 9). Each generation in the pedigree shows the sire and dam chosen for breeding purposes together with their female sibs.

None of the birds used was inoculated. Except for those males used for breeding purposes the data consist of females only. Necropsies were performed on all birds over 30 days of age and diagnoses were based on gross necropsy findings only. All chickens in the four latest generations were held until they were 600 days of age at which time they were killed and examined. Some of the chickens in the first three generations were held longer than the 600-day period.

All of the chickens in this study, both resistant and susceptible, were incubated

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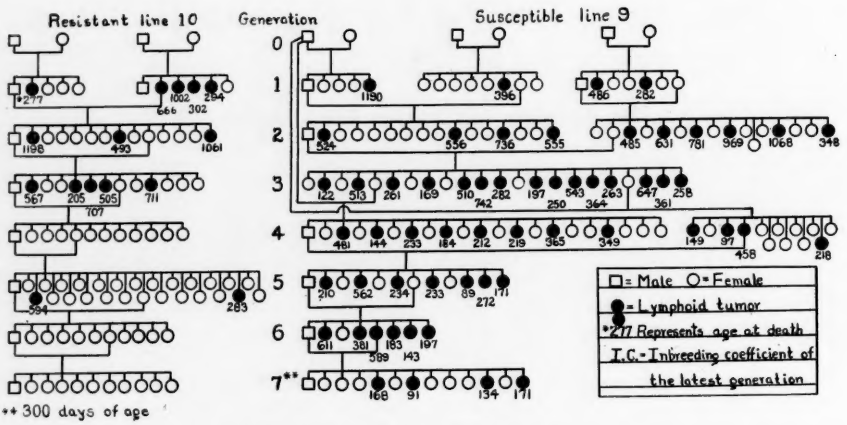

RESISTANT AND SUSCEPTIBLE LINES

Figure 9

Pedigrees showing the presence of lymphoid tumors among resistant (I.C. 73 per cent) and susceptible (I.C. 50 per cent) inbred chickens.

and hatched together. Further, the chickens were reared as a mixed population throughout the entire experimental period. Thus, all chickens in a given population were exposed, more or less equally, to whatever level of exposure existed.

The term "line" as used in this text refers to a group of birds with a relatively high *inter se* relationship between sibs or near relatives resulting from restricted ancestry. The term "family" refers to progeny of a single sire and dam.

The history of these chickens, together with the general management practices followed, has been previously reported by Waters.[8,9]

Description of Pedigrees and Results

Figure 9 shows the pedigrees of certain families from susceptible line 9 and resistant line 10 developed by Waters and Prickett,[11] together with the incidence of lymphoid tumors and the age at which these tumors were observed. The original foundation chickens shown in the pedigree and designated as the 0 generation were not reared at this Laboratory but represent the parents from which eggs were obtained to provide the progeny shown in generation 1. The parents in all succeeding generations were bred at about 250 to 300 days of age. In general, the choice of breeding birds was based on the incidence of tumors, together with the age at which such tumors occur, among their sibs and their dam's sibs. For the first three generations it was possible to hold a portion of the birds for as many as 1,400 days. In the four later generations space limitations necessitated the sacrificing of all birds at 600 days of age. A study of the pedigrees during the early generations will show that a number of birds had tumors after 600 days of age. However, it is not possible from these data to make comparisons after 600 days between the two groups for all generations inasmuch as the last four generations were all sacrificed at 600 days of age.

A comparison of the chickens in the two pedigrees at 600 days of age shows that for the first two generations there is no significant difference in the incidence of tumors between the resistant and susceptible groups. Beginning with the third generation and in all subsequent generations there is a marked dif-

ference between the chickens in the two pedigrees. Among the resistant birds at 600 days of age for all generations, 10 per cent show tumors as contrasted with 37 per cent for the susceptible chickens. Numerous deaths occurred in both lines which could not be attributed to the presence of tumors. Presumably some of these chickens would have had tumors had they lived. However, losses which could not be attributed to lymphomatosis were about the same in both pedigrees, therefore, such losses should not perceptibly bias the results.

The data in these two pedigrees show that genetic selection with inbreeding will reduce the incidence of lymphoid tumors in certain families while in other families the incidence of such tumors is increased within a given experimental period. Thus, breeding for resistance to lymphomatosis in chickens would appear to be a useful method of decreasing the incidence of this disease.

Discussion

Research at this Laboratory makes it increasingly evident that lymphomatosis is a contagious and malignant disease of chickens characterized by the presence of lymphoid tumors which may occur in almost any tissue of a bird's body. In this report it has been demonstrated that selection with intense inbreeding can, within a few generations, segregate groups of chickens which are resistant to, or susceptible to, naturally occurring lymphomatosis. However, before resistant or susceptible individuals can express their genetic possibilities, they must be exposed to the agent or agents responsible for the disease causing the tumors. Waters[9] and Waters and Prickett[11] have shown that a fairly high average incidence of lymphomatosis was present in each of the mixed populations to which the chickens in the two pedigrees were exposed. Waters and Prickett[10] and Waters[8] demonstrated that susceptible chickens rarely develop tumors if they are maintained in an environment "free" of the disease. One other known variable of considerable importance pertains to the egg as a carrier of this disease.[10, 8]

Previously there has been no clear oncological evidence which would indicate that the causative agent of tumors is contagious in nature. This raises the question as to whether the lymphoid tumors which characterize this disease in chickens represent neoplastic growth or whether they are of the order of a granulomatous lesion.

Summary

Two pedigrees are given showing the incidence of lymphoid tumors, characterizing lymphomatosis, occurring within resistant and susceptible families of inbred White Leghorn chickens.

The data, collected over a period of seven years, show that genetic selection has reduced the incidence of tumors in certain families, while in other families the incidence of tumors has increased.

Evidence has been presented to show that breeding for resistance to lymphomatosis in chickens is a useful and practical method for decreasing the incidence of this disease.

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AN UNUSUAL GROWTH OF BONE IN CHICKENS

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NEW BONE STRUCTURE
Figure 10

The rather elaborate process characterizing the tip of the sternum on the left is unique, on the basis of an examination of a very large number of specimens. It appeared in two related chickens. Such a rare variation, if genetically conditioned, might mark an important step in evolution.

AN experiment now in progress at the Kentucky Agricultural Experiment Station concerns the comparative rate of growth and comparative rate of calcification of the larger bones of male and female chickens with and without crooked keels.

The large bones of 150 New Hampshire chickens have been prepared for anatomical study. One of the authors (Wachs) while making such study of the dried bones observed an anomalous unilateral condition in one 12-week old pullet (1420 grams) and in one cockerel (1540 grams) of the same age. The anomaly consisted of a bony arch or connection near the proximal ends of the right lateral internal and right lateral external processes of the sternum. The accompanying picture compares this condition with a normal sternum of a pullet of the same breed and age. In proportions and texture this extra bone connection was similar to the two lateral proceses of the sternum which it joined. The relationship of the two chicks having this condition is not known, but it is known that they came from the same flock of hens and were the same age. This is the first time that this unusual growth of bone has been observed at this Station. A survey of available literature did not disclose other occurrences of this anomaly.

While it is impossible to give the true reason why this unusual bone formation occurred, yet it may be proper to infer or reason that at some early stage in the development of the domestic fowl such linkage of these two lateral processes may have been normal. That the connection adds materially to the strength of the processes is quite apparent.

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*The investigation reported in this paper is in connection with a project of the Kentucky Experiment Station and is published by permission of the Director.

INHERITANCE OF ZYGODACTYLY

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WEBBING, or zygodactyly, of the second and third toes is not a very uncommon human anomaly. Webbing of the third and fourth fingers is rarer but, as shown by Cummins,[3] the occurrence of an interdigital triradius, which marks the site of the webbing, can be present even though the webbing is barely noticeable. In zygodactyly there is no osseous malformation, like that found in syndactyly[7] or polydactyly. The character is not easy to identify in cases where the deviation from the normal is slight and, in most cases, is only visible on the feet. It is found more than twice as frequently in males as in females though it is conceivable that this difference may be partly accounted for by a greater reticence on the part of females concerning their toes.

Striking Irregularity of Transmission

Pedigrees have been reported, in which the condition shows an irregular dominance,[5,6] but some pedigrees show other peculiar features. Schofield[10] described a family wherein transmission from fathers to all their sons and to none of their daughters occurred in three generations and this pedigree has been generally accepted as a demonstration of *Y*-chromosome inheritance in man.[2] However, other pedigrees, notably those of Guyer,[4] Stiles and Hawkins,[12] suggest the possibility of sex-linked *X*-chromosome inheritance. Pipkin and Pipkin advocate the hypothesis of partial sex-linkage to explain the incidence in pedigrees.[9] However, irregular dominance[14] coupled with a predisposition for males rather than females to express the character would be an adequate explanation for all the pedigrees, were it not for one peculiar feature. The incidence of the condition among the children of affected females is only about one quarter of the incidence among children of affected males. This is shown in Table I, where the children of affected subjects are listed.

Without making any allowance for the imperfectly specified personnel of the Schultz pedigrees (ii) and (iii), there are 69 affected offspring against 40 normal of affected fathers, whereas affected mothers only show 11 affected offspring against 27 normal (Table II). The difference between the two ratios is obviously significant ($\chi^2=13.41$) and would have been increased by the inclusion of

TABLE I.—Summary of Zygodactylous Inheritance Reported by Nine Authors

Source	Offspring of Affected Fathers				Offspring of Affected Mothers			
	Affected		Normal		Affected		Normal	
	♂	♀	♂	♀	♂	♀	♂	♀
Hurlin[6]	3	1	1	2	..	..	..	..
Schofield[10]	13	..	..	11	..	..	..	..
Schultz[11] (i)	9	5	..	6	1	..	..	4
(ii)	7	3	1	..	2	1	1*	2*
(iii)	8†	6	..	..	..	..	2‡	1
Wright[14]	1	2	1	2	..	2	..	..
Castle[1]	..	..	..	..	..	3	4	3
Guyer[4]	..	..	2	2	..	..	..	..
Heston[5] (i)	1	1	..	..	4	..	2	5
(ii)	3	..	2	1	..	..	..	..
Pipkin & Pipkin[9] (i)	1	..	2	3	..	..	..	..
(ii)	3	..	..	4	..	..	..	..
Stiles & Hawkins[12]	..	2	..	..	..	..	3	..
Total	49	20	9	31	7	4	12	15

*There were 5 unaffected also, with sexes unspecified.
†There were possibly other affected sons.
‡There were possibly other normal sons.

five offspring with sexes unspecified and one or more extra unaffected sons of affected mothers together with one or more affected sons of affected fathers from the Schultz pedigrees.

Theory of Antigenic Incompatability

It is difficult to imagine any simple genetical cause, which would account for predominance of paternal hereditary influence which applies to offspring of both sexes. The suggestion can, however, be made that zygodactyly is due to mild antigenic incompatability of mother and foetus. Any particular incompatible gene responsible for such a situation must be inherited from the father. If more than one antigen can give rise to the same effect (as appears to be the case in erythroblastosis foetalis) the mother can occasionally be affected as well as her child. The present writer has recently suggested that foetal antigenic influences, to which the maternal tissues react very early in the period of gestation (second and third months), may be the main cause of a group of abnormalities of foetal growth including mongolism.[8] Such antigenic genes would have to be assumed to be very prevalent and their presence would not always cause disturbances of foetal growth. Zygodactyly, which, incidentally, is not uncommonly found in mongoloid dwarfs,[13] may itself be due to antigenic incompatibility of mother and foetus.

To complete the picture, the offspring of unaffected members of these pedigrees need to be studied. On the traditional hypothesis of irregular dominance, these unaffected parents would be termed "transmitters." On the hypothesis of antigenic incompatibility the term "transmitter," however, loses its usual meaning. In Table III, the incidence of zygodactyly among the offspring of unaffected fathers and unaffected mothers is compared. The numbers are small but they show a tendency which is the reverse of that shown in Table I. The proportions of sons affected in both cases again are greater than the proportions of daughters affected but the incidence of zygodactyly is more marked in the children of the unaffected mothers than in the children of unaffected fathers.

The effect can be summarized by saying that the correlation of father and child with respect to the trait is significantly greater than the correlation of mother and child. This is typical for a trait due to inherited foetal incompatibility. The correlations, as shown in Table IV, are both much lower than those which would be found in the general population for the trait concerned since the proportionate number of families, in which parents and children are all unaffected, are excluded and many

TABLE II.—Summary of Offspring of Affected Fathers and Mothers

Offspring	Fathers	Mothers	Total
Affected	69	11	80
Normal	40	27	67
Total	109	38	147

$$\chi^2 = \frac{[(69\times27)-(11\times40)]^2\times147}{80\times67\times109\times38} = 13.41.$$

TABLE III.—Distribution of Children of Normal Parent

Normal Parent	Source	Affected ♂	Affected ♀	Normal ♂	Normal ♀
Father	Wright[14]	..	1	..	..
	Heston[5](i)	2	2	1	1
	(ii)	2	..	3	9
	Pipkin & Pipkin[9](i)	1	..	..	2
	Stiles & Hawkins[12]	..	1	..	2
	Total	5	4	4	14
Mother	Heston[5](ii)	1	1	..	..
	Guyer[4]	2	..	..	1
	Total	3	1	..	1

TABLE IV.—Correlations of Parents and Offspring

Offspring of Fathers	Affected	Normal	Total
Affected	69	9	78
Normal	40	18	58
Total	109	27	136

$$r_f = \frac{(69\times18)-(40\times9)}{\sqrt{(78\times58\times109\times27)}} = +0.242$$

Offspring of Mothers	Affected	Normal	Total
Affected	11	4	15
Normal	27	1	28
Total	38	5	43

$$r_m = \frac{(11\times1)-(27\times4)}{\sqrt{(15\times28\times38\times5)}} = -0.363$$

$$r_f - r_m = 0.605 \pm 0.175$$

cases also in which both parents are normal and some children affected. However, the comparison between mothers and fathers is not thereby invalidated.

Summary

The material from published pedigrees of zygodactyly has been analyzed and it has been shown that paternal hereditary influence predominates over maternal. The phenomenon is interpreted as evidence that the trait is primarily due to incompatibility of the mother with some unknown foetal antigen inherited from the father.

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A PEDIGREE OF RHEUMATOID SPONDYLITIS

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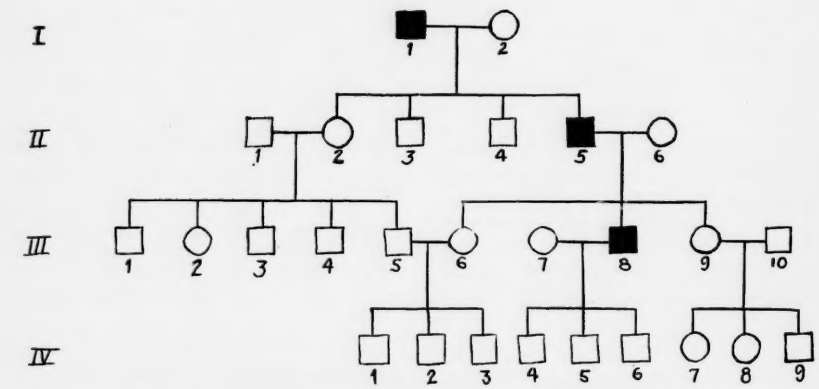
Rheumatoid spondylitis is defined in a recent paper by Boland and Present[1] as a "chronic progressive disease of the spine, the fundamental lesions of which occur in the diarthrodial or synovial joints." The disease usually begins in the sacro-iliac joints and may spread to the synovial joints of the lumbar, thoracic and cervical regions, resulting in complete ankylosis of the spine and sometimes accompanied by an involvement of the hips, knees, shoulders, elbows and hands and feet. With its more severe expression there is diminished chest expansion, lumbodorsal stoop and various constitutional reactions. However it may become arrested at any point and be characterized by relatively mild symptoms. One of the most constant symptoms is an elevated sedimentation rate. In 78 of 100 cases of soldiers suffering from this disease in an army general hospital the rate was above normal. In a study of these 100 cases no precipitating cause such as infection, injury, exposure or psychic strain could be found in 80 per cent of the individuals and in the other 20 per cent there were various possible contributing causes, none of which could be regarded as solely responsible. Unfortunately no studies of the incidence of the disease in the families of these cases was mentioned. Moreover in a search through the literature the authors have been unable to find any papers dealing with the genetics of this disorder.

Various synonyms for the disease are in use, resulting in considerable confusion in terminology. These include Marie-Strumpell's disease, spondylitis chronica ankylopoietica, von Bechterew's syndrome, spondylarthritis and others. These reflect the difficulty of recognizing a group of symptoms as belonging to a clear-cut disease entity.

In the family under consideration in this paper there is direct transmission of the disease by males through three generations (Figure 11). The data for each case follows:

I-1.—He was known to suffer from ankylosis of the lower spine. He carried with him, wherever he travelled, a specially designed chair. Died at 73 from a stroke.

II-5.—Spondylitis began in his early thirties and progressed until his neck was com-



MALE TRANSMISSION OF SPINAL RHEUMATISM

Figure 11

pletely "set up." Apparently his fingers were also affected as there was difficulty in moving them. Died at 45 of heart attack.

III-8.—He has had trouble with his back from an early age. Symptoms at onset were a stiffening of the lower spinal region accompanied by considerable pain. The joints would set up and loosen again. Later the ankylosis progressed to the neck, followed shortly by a stiffening of the shoulder joints (bursitis). Many of the joints ache and crack. Herberden's nodes are present on the distal digital joints and calcification nodes on the palms. Additional symptoms include eczema of the scalp during severest pain, hives and constipation. The condition was diagnosed several years ago by orthopedic surgeons as Marie-Strumpell's disease. About a year ago he nearly lost consciousness as a result of strenuous physical exertion. Taken to a hospital he was found to be suffering from diabetes. At present when his insulin and food are balanced the pain from his other condition appears considerably lessened.

From the information available concerning I-1 and II-5 it appears likely that these individuals had the same disease that III-8 has, although at the time when they lived such a diagnosis would hardly have been possible. It should also be noted that the disease may still make its appearance in the fourth generation since the sons of III-8 are all well under thirty years of age. Of the 100 cases previously mentioned 15 had their onset between the ages of 16 and 19, 69 between 20 and 29 and 16 between 30 and 39.

On the basis of the appearance of a trait in three successive generations one is not justified in concluding that heredity is the chief variable in its expression. However, in view of the relative infrequency of this disease in the general population it is extremely unlikely that chance alone is responsible for its incidence in this family. It would seem that genic action is one of the important causes of the disease in this family, and a dominant gene appears to be involved, with low penetrance in females. In view of the fact that males are affected much more frequently than females, the ratio being about 20:1, it is possible that inheritance is sex-influenced. Reliable data showing the incidence of the condition in the general population were not available for gene-frequency analysis. It is evident, however, why family cases of the disease might not be detected very often. When transmission is through females one or more generations might be skipped. Again, the expressivity might be so low in some cases that it would escape detection. However, this does not preclude the possibility that many cases of the disease may result almost wholly from extrinsic factors.

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1. Boland, E. W., and A. J. Present. *Jour. Amer. Med. Assoc.* 129:843-849. 1945.

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